AMENDMENTS TO THE CLAIMS

The claims in this listing will replace all prior versions, and listings, of claims in the application. Please amend the claims as follows.

Listing of Claims:

1. (Original) A method for determining an inflammatory disease, which comprises detecting at least one gene polymorphism existing in at least one gene selected from the group consisting of a lymphotoxin-α (LT-α) gene, an I Kappa B-like (IKBL) gene, and a BAT1 gene.

2. (Original) A method for determining an inflammatory disease, which comprises detecting at least one single nucleotide polymorphism existing in at least one gene selected from the group consisting of the lymphotoxin-α (LT-α) gene, the I Kappa B-like (IKBL) gene, and the BAT1 gene.

3. (Currently Amended) A method for determining an inflammatory disease, which comprises detecting at least one single nucleotide polymorphism selected from the group consisting of the following (1) to (5):

(1) a G/A polymorphism at nucleotide 10 in the nucleotide sequence of exon 1 of the LT-α gene shown in SEQ ID NO: 1;

(2) an A/G polymorphism at nucleotide 90 in the nucleotide sequence of intron 1 of the LT-α gene shown in SEQ ID NO: 2;

(3) a C/A polymorphism at nucleotide ~~81~~ 80 in the nucleotide sequence of exon 3 of the LT-α gene shown in SEQ ID NO: 3;

(4) a T/A polymorphism at nucleotide 572 in the nucleotide sequence of a promoter of the IKBL gene shown in SEQ ID NO: 4; and

(5) a G/C polymorphism at nucleotide 1228 in the nucleotide sequence of a promoter of the BAT1 gene shown in SEQ ID NO: 5.

4. (Original) A method for determining an inflammatory disease, which comprises detecting whether or not the combination of the nucleotide 10 in the nucleotide

sequence of exon 1 of the LT-α gene shown in SEQ ID NO: 1 and the nucleotide 90 in the nucleotide sequence of intron 1 of the LT-α gene shown in SEQ ID NO: 2 is G-A heterozygote, A-G heterozygote or A-A homozygote.

5. (Original) A method for determining an inflammatory disease, which comprises detecting a gene polymorphism whereby an amino acid to be encoded is mutated from threonine to asparagine by substitution of at least one of the nucleotides 80 to 82 in the nucleotide sequence of exon 3 of the LT-α gene shown in SEQ ID NO: 3 with another nucleotide.

6. (Previously Amended) The method according to claim 1 wherein the inflammatory disease is myocardial infarction.

7. (Currently Amended) The method according to claim 1, which comprises an oligonucleotide as a probe that can hybridize to a sequence of at least 10 continuous nucleotides containing at least one position selected from the group consisting of the following (1) to (5), the position being contained in the sequences shown in SEQ ID NOS: 1 to 5, or to a complementary sequence thereof:

(1) position 10 of the nucleotide sequence of exon 1 of the LT-α gene shown in SEQ ID NO: 1;

(2) position 90 of the nucleotide sequence of intron 1 of the LT-α gene shown in SEQ ID NO: 2;

(3) position ~~81~~ 80 of the nucleotide sequence of exon 3 of the LT-α gene shown in SEQ ID NO: 3;

(4) position 572 of the nucleotide sequence of the promoter of the IKBL gene shown in SEQ ID NO: 4; and

(5) position 1228 of the nucleotide sequence of the promoter of the BAT1 gene shown in SEQ ID NO: 5.

8. (Currently Amended) The method according to claim 1, which comprises an oligonucleotide as a primer that can amplify a sequence of at least 10 continuous

nucleotides containing at least one position selected from the group consisting of the following (1) to (5), the position being contained in the sequences shown in SEQ ID NOS: 1 to 5, and/or to a complementary sequence thereof:

(1) position 10 of the nucleotide sequence of exon 1 of the LT-α gene shown in SEQ ID NO: 1;

(2) position 90 of the nucleotide sequence of intron 1 of the LT-α gene shown in SEQ ID NO: 2;

(3) position ~~81~~ 80 of the nucleotide sequence of exon 3 of the LT-α gene shown in SEQ ID NO: 3;

(4) position 572 of the nucleotide sequence of the promoter of the IKBL gene shown in SEQ ID NO: 4; and

(5) position 1228 of the nucleotide sequence of the promoter of the BAT1 gene shown in SEQ ID NO: 5.

9. (Previously Amended) The method according to claim 8 wherein the primer is forward primer and/or reverse primer.

10. (Currently Amended) A kit for diagnosing an inflammatory disease, which comprises at least one oligonucleotide that can (a) hybridize to, (b) amplify, or (c) hybridize to and amplify a sequence of at least 10 continuous nucleotides containing at least one position selected from the group consisting of the following (1) to (5), the position being contained in the sequences shown in SEQ ID NOS: 1 to 5, or to a complementary sequence thereof:

(1) position 10 of the nucleotide sequence of exon 1 of the LT-α gene shown in SEQ ID NO: 1;

(2) position 90 of the nucleotide sequence of intron 1 of the LT-α gene shown in SEQ ID NO: 2;

(3) position ~~81~~ 80 of the nucleotide sequence of exon 3 of the LT-α gene shown in SEQ ID NO: 3;

(4) position 572 of the nucleotide sequence of the promoter of the IKBL gene shown in SEQ ID NO: 4; and

(5) position 1228 of the nucleotide sequence of the promoter of the BAT1 gene shown in SEQ ID NO: 5.

11. (Original) The kit according to claim 10 wherein the inflammatory disease is myocardial infarction.

12. (Currently Amended) A method for analyzing the expression state of LT-α, IKBL, or BAT1, which comprises detecting at least one single nucleotide polymorphism selected from the group consisting of the following (1) to (5):
(1) a G/A polymorphism at nucleotide 10 in the nucleotide sequence of exon 1 of the LT-α gene shown in SEQ ID NO: 1;
(2) an A/G polymorphism at nucleotide 90 in the nucleotide sequence of intron 1 of the LT-α gene shown in SEQ ID NO: 2;
(3) a C/A polymorphism at nucleotide ~~81~~ 80 in the nucleotide sequence of exon 3 of the LT-α gene shown in SEQ ID NO: 3;
(4) a T/A polymorphism at nucleotide 572 in the nucleotide sequence of a promoter of the IKBL gene shown in SEQ ID NO: 4; and
(5) a G/C polymorphism at nucleotide 1228 in the nucleotide sequence of a promoter of the BAT1 gene shown in SEQ ID NO: 5.

13. (Currently Amended) A method for measuring the transcriptional activity of LT-α, IKBL, or BAT1, which comprises introducing an LT-α, IKBL, or BAT1 gene fragment containing at least one single nucleotide polymorphism selected from the group consisting of the following (1) to (5) into a cell, culturing the cell, and analyzing the expression of the gene:
(1) a G/A polymorphism at nucleotide 10 in the nucleotide sequence of exon 1 of the LT-α gene shown in SEQ ID NO: 1;
(2) an A/G polymorphism at nucleotide 90 in the nucleotide sequence of intron 1 of the LT-α gene shown in SEQ ID NO: 2;
(3) a C/A polymorphism at nucleotide ~~81~~ 80 in the nucleotide sequence of exon 3 of the LT-α gene shown in SEQ ID NO: 3;

(4) a T/A polymorphism at nucleotide 572 in the nucleotide sequence of a promoter of the IKBL gene shown in SEQ ID NO: 4; and
(5) a G/C polymorphism at nucleotide 1228 in the nucleotide sequence of a promoter of the BAT1 gene shown in SEQ ID NO: 5.

14. (Currently Amended) A method for screening for a substance inhibiting the transcriptional activity of LT-α, IKBL, or BAT1, which comprises introducing an LT-α, IKBL, or BAT1 gene fragment containing at least one single nucleotide polymorphism selected from the group consisting of the following (1) to (5) into a cell, culturing the cell in the presence of a candidate substance inhibiting the transcriptional activity of LT-α, IKBL, or BAT1, and analyzing the expression of the gene:
(1) a G/A polymorphism at nucleotide 10 in the nucleotide sequence of exon 1 of the LT-α gene shown in SEQ ID NO: 1;
(2) an A/G polymorphism at nucleotide 90 in the nucleotide sequence of intron 1 of the LT-α gene shown in SEQ ID NO: 2;
(3) a C/A polymorphism at nucleotide ~~81~~ 80 in the nucleotide sequence of exon 3 of the LT-α gene shown in SEQ ID NO: 3;
(4) a T/A polymorphism at nucleotide 572 in the nucleotide sequence of a promoter of the IKBL gene shown in SEQ ID NO: 4; and
(5) a G/C polymorphism at nucleotide 1228 in the nucleotide sequence of a promoter of the BAT1 gene shown in SEQ ID NO: 5.

15. (Original) A substance inhibiting the transcriptional activity of LT-α, IKBL, or BAT1, which is obtained by the screening method of claim 14.

16. (Previously Amended) The method according to claim 13, which comprises introducing a transcriptional unit wherein a reporter gene is ligated downstream of said LT-α, IKBL, or BAT1 gene fragment into a cell, culturing the cell, and analyzing the expression of the gene by measuring the reporter activity.

17. (Original) The method according to claim 16, wherein said reporter gene is luciferase gene.

18. (Currently Amended) A method for screening for a transcriptional regulatory factor of LT-α, IKBL, or BAT1, which comprises bringing a gene fragment containing at least one single nucleotide polymorphism selected from the group consisting of the following (1) to (5) into contact with a sample wherein a transcriptional regulatory factor of LT-α, IKBL, or BAT1 is presumed to be present, and detecting binding of the above fragment with the transcriptional regulatory factor:
(1) a G/A polymorphism at nucleotide 10 in the nucleotide sequence of exon 1 of the LT-α gene shown in SEQ ID NO: 1;
(2) an A/G polymorphism at nucleotide 90 in the nucleotide sequence of intron 1 of the LT-α gene shown in SEQ ID NO: 2;
(3) a C/A polymorphism at nucleotide ~~81~~ 80 in the nucleotide sequence of exon 3 of the LT-α gene shown in SEQ ID NO: 3;
(4) a T/A polymorphism at nucleotide 572 in the nucleotide sequence of a promoter of the IKBL gene shown in SEQ ID NO: 4; and
(5) a G/C polymorphism at nucleotide 1228 in the nucleotide sequence of a promoter of the BAT1 gene shown in SEQ ID NO: 5.

19. (Original) The method according to claim 18 wherein the detection is carried out by gel shift assay.

20. (Original) A method for evaluating ability to induce an adhesion molecule in a cell, which comprises introducing a gene fragment containing a C/A polymorphism at nucleotide 90 in a nucleotide sequence of intron 1 of an LT-α gene shown in SEQ ID NO: 2 into a cell in which an adhesion molecule can be induced, and evaluating the ability to induce an adhesion molecule in the cell.

21. (Original) The method according to claim 20 wherein said cell is human coronary-artery smooth-muscle cell (HCASMC).

22. (Original) The method according to claim 20 wherein said adhesion molecule is vascular cell-adhesion molecule-1 (VCAM-1) or E-selectin.

23. (Original) A method for treating an inflammatory disease, which comprises suppressing the expression or activity of lymphotoxin-α (LT-α).

24. (Original) The method according to claim 23 wherein the inflammatory disease is myocardial infarction.

25. (Previously Amended) The method according to claim 23 wherein an antibody against lymphotoxin-α (LT-α) is used.

26. (Original) A therapeutic agent for an inflammatory disease, which comprises as an active ingredient a substance suppressing the expression or activity of lymphotoxin-α (LT-α).

27. (Original) The therapeutic agent according to claim 26 wherein the substance suppressing the expression or activity of lymphotoxin-α (LT-α) is an antibody against lymphotoxin-α.

28. (Previously Amended) A method for screening for a therapeutic agent for an inflammatory disease, which comprises contacting a cell with a candidate substance, analyzing the expression level of a gene encoding lymphotoxin-α (LT-α) within the cell, and selecting as a therapeutic agent for an inflammatory disease a candidate substance that lowers the expression level of the gene by comparison with a condition where the candidate substance is absent.

29. (Previously Amended) A method for screening for a therapeutic agent for an inflammatory disease, which comprises contacting lymphotoxin-α (LT-α) with a candidate substance, measuring the activity of lymphotoxin-α, and selecting as a

therapeutic agent for an inflammatory disease a candidate substance that lowers the activity of lymphotoxin-α by comparison with a condition where the candidate substance is absent.

30. (Original) The method according to claim 29 wherein the activity of lymphotoxin-α is an activity to induce an adhesion molecule and/or a cytokine.

31. (Original) The method according to claim 30 wherein the adhesion molecule is VCAM-1, ICAM-1, or E-selectin, and the cytokine is TNF.

32. (Previously Amended) The method according to claim 28 wherein the expression level or activity of lymphotoxin-α (LT-α) is lowered through an increase in the expression level or activity of the IKBL gene.